REMARKS

The abstract is objected to due the informality. Informalities pointed out by the Examiner have been corrected on abstract.

Claims 14-16 and 18-25 are rejected under U.S.C. 112.

Lan Xiang Xi is a **pure ingredient** extracted from plant, which has Latin genus-species name: *Curcuma aromatica Salisb.* Lan Xiang Xi is extracted from root tuber or whole plant of *Curcuma aromatica Salisb.* Root tuber has higher concentration of LX. However, different plants of whole plant have LX too.

Further on, "Lan Xiang Xi (LX)" is not a name of plant. It is a name of one ingredient extracted from *Curcuma aromatica Salisb* .For example, on page 4, line 10-18 described chemistry data of LX as the following.

Molecular form: $C_{15}H_{24}$

Molecular weight: 204

Mp: 114~118°C

$[\alpha]^{16}$: -15°

IRv^{KBr} cm^{-1}: 3090, 2975, 2860, 1642, 1440, 1375, 1002, 910, 888 ($C=CH_2$);

PMR (CCl_4) δ: 0. 97 (3H, s), 1.7 (6H, s), 4.4~5.6 (6H, m), 5.7 (1H, dd, $=OH_2$);

MSm/e (%): 204 (M^+), 147 (33), 121 (41), 107 (54), 93 (89), 81 (100), 79 (44), 68 (74), 67 (52), 55 (41), 53 (33), 41(52).

"One kg of plant powder" means one kg powder of *Curcuma aromatica Salisb* or one kg powder of root tuber of *Curcuma aromatica Salisb.*

Claim 15 has informal words therefore this claim was rewritten. Claim 18 was cancelled.

As experimental data indicated that DGL and LXL have synergetic pharmaceutical action, but only one ingredient (LX or LXL) still has some function too. Of course, LX + PDG is better than single ingredient (LX). For example, as Table 1 showed below:

Table 1. The effect of LX and PDG concentration on inhibition of oncogenes

LX concentration (ng/ml)	Inhibition (%)		
	c-myb RNA	c-myc RNA	RAS
Saline	0	0	0
LX (10)	25.0 ± 2.8*	24.2 ± 2.8*	15.2 ± 2.0*
LX (50)	68.5 ± 7.8**	78.0 ± 8.5**	28.2 ± 3.8**
LX (50) + PDG (50)	92.2 ± 12.50***	90.2 ± 10.5**	45.8 ± 6.2

As data of Table 1 showed that LX could inhibit c-myb RNA. When the concentration of LX was 50 ng/ml, its inhibition of c-myb RNA was 68.5%. When concentration of LX was 50 ng/ml and PDG was 50 ng/ml LX plus PDG have stronger effects, for example, its inhibition rate was rose to 92.2%. It was described (line 9 from bottom of page 7) that "This study clearly indicated that LX and LX + PDG could significantly inhibit oncogenes of cancer cells."

In the same way, inhibition of c-myc RNA by 50 (ng/ml) of LX is 78.0% but inhibition of LX (50 ng/ml) + PDG (50 ng/ml) is 90.2%. For RAS, inhibition of LX (50 ng/ml) is 28.2% but inhibition of LX (50 ng/ml) + PDG (50 ng/ml) is 45.8%.

The results for inducing differentiation of cancer cells by LX and LX + PDG have sane situation (see table 3).

Table 3. Induced differentiation of LX and PDG on cancer cells

Treatment	NBT%*
None	0
LX	68.5 ± 7.2
LX + PDG	72.8 ± 8.5

As data of Table 3 indicated, for single LX, NBT% is 68.5%, but for LX + PDG, its NBT% is 78.8%.

Same results can be seen in Table 4.

Table 4. Effect of LX and PDG on inhibiting growth cancer cells

Cell line	Inhibition (%)	
	LX	LX + PDG
Control	---	---
Human cells		
HL-60	78.5 ± 8.2	82.8 ± 9.0
Hela	70.7 ± 8.0	84.0 ± 9.8
KB	73.8 ± 8.9	80.8 ± 7.8

The data of Table 4 indicated that inhibition (%) of LX to HL-60 is 78.5, but LX + PDG is 82.8. The results of Hela and KB are as same as HL-60.

The results of inducing apoptosis, inhibiting transplanted tumors and tumor incidence have same situation. It indicated that single LX could induce apoptosis and inhibit transplanted tumors and tumor incidence. But LX plus PDG has stronger pharmaceutical function.

Claims 18-25 were rewritten and have more limits.

Examiner attached "Notice of References Cited" including art of Weng X.C. and Feng, J. et al. Now new claims withdraw items regarding increasing immune function.

All new claims have been rewritten to include more limitations.

It is therefore respectfully submitted that this application is in condition for allowance and should be allowed forthwith. An early notice to this effect is solicited.

Respectfully submitted

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